

Models of Urinary Acidification

by

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Submitted to the Department of Chemical Engineering
on February 12, 1982 in partial fulfillment of
the requirements for the degree of
Master of Science in Chemical Engineering

Abstract

The model of proximal tubule bicarbonate reabsorption developed by Wang and Deen has been modified to include: (1) axial variation of intracellular pH; (2) hydrogen secretion kinetics of the form $J_H = P_H (\alpha C_{4C} - C_{4L})$, where P_H is apparent H^+ permeability, α is a measure of the secretion driving force and C_{4C} and C_{4L} are the intracellular and luminal hydronium concentrations; (3) a paracellular bicarbonate leak from the interstitial fluids to the tubule lumen; and (4) ammonia and phosphate buffers.

The model predicts the intracellular pH to be higher than luminal pH everywhere except in the initial portion of the proximal tubule. Hydrogen secretion decreases with increasing luminal acidity. The ammonia and phosphate buffer systems and the paracellular bicarbonate leak result in higher luminal pH in the distal tubule segments. The paracellular leak reaches 12-15% of the bicarbonate efflux into the interstitial fluid by the proximal tubule end. By increasing the paracellular permeability coefficient, a steady state luminal pH is attained. Reabsorption rates of tCO_2 of 120-140 μ moles/min/mm are predicted. Fractional reabsorption of phosphate is greater than 70%. The luminal concentration of monovalent phosphate increases to levels higher than plasma values so that passive transport of monovalent phosphate is sustained over the length of the proximal tubule. Total ammonia content increases to .4mM by the tubule end. A 50% increase in SNGFR results in a 7-23% increase in absolute tCO_2 reabsorption but a 30-33% decrease in fractional tCO_2 .

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